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FALL IN IgE LEVELS AFTER TREATMENT FOR HOOKWORM

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SUMMARY

Serum IgE levels were high in a Papua New Guinean population infested with hookworm. Serum IgE and blood eosinophil levels fell after treatment with anthelmintics.

INTRODUCTION

Raised serum levels of immunoglobulin E (IgE) have been found in a variety of helminth infestations including *Ascaris lumbricoides* (Johansson, Mellbin & Vahlquist, 1968), *Toxocara* spp. (Hogarth-Scott, Johansson & Bennich, 1969), *Trichinella spiralis* (Rosenberg, Polmar & Whalen, 1971), *Schistosoma japonicum*, *Wuchereria bancrofti* and hookworm (Ito, Sawada & Sato, 1972).

Biroum-Noerjasin (1973) reported increased serum IgE levels after treatment for hookworm. This is in contrast to the fall which occurred after treatment in patients with intestinal capillariasis (Rosenberg *et al.*, 1970), and 6 months after an acute episode of trichinosis (Rosenberg *et al.*, 1971). This study was undertaken to determine whether the findings of Biroum-Noerjasin could be repeated.

MATERIALS AND METHODS

The investigation was carried out in the Goroka Subdistrict of the Eastern Highlands District of Papua New Guinea. Subjects were members of two villages located at an altitude of 1700 metres. The average rainfall is 80 inches per annum with partially defined wet and dry seasons. Almost all the villagers were unshod. Although some villages had latrines, they were used infrequently and often were in a state of disrepair. Defaecation occurred in the bush, vegetable gardens or coffee plantations.

Faeces were collected in airtight plastic containers and faecal egg counts measured by the

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method of Gordon & Whitlock (1939). Six specimens were cultured by the method of Harada & Mori (1955) and identified as *Necator americanus*.

Serum IgE levels were determined by the radioactive single radial diffusion method (Rowe, 1969). The standard was from a batch of pooled human serum (69/204) supplied by the World Health Organization. Blood eosinophil levels were measured by the counting chamber method described by Dacie (1968) using eosin–acetone diluting fluid and a Neubauer counting chamber.

Faecal and blood samples were collected in fifty-three subjects who were then given a single dose of 900 mg of Pyrantel Embonate ('Combantrin', G.P. Laboratories). Blood samples were collected 6 weeks later in all subjects and faecal specimens in twenty-seven subjects. Another five subjects were given Pyrantel Embonate, 600 mg weekly for 6 weeks, and blood and faecal specimens collected fortnightly for 12 weeks. Control subjects were given 25 mg of vitamin C.

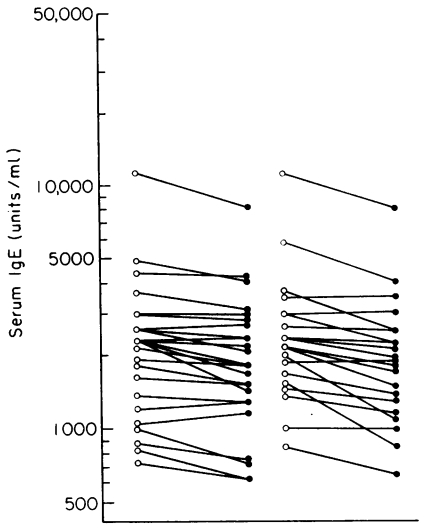

FIG. 1. Serum IgE levels before and 6 weeks after anthelmintic treatment in forty-six subjects. (○) Pre-treatment levels. (●) Post-treatment levels.

RESULTS

Hookworm ova were detected in samples from all subjects. Twenty-one per cent had egg counts of less than 1000, 43% between 1000 and 5000, 21% between 5000 and 10,000 and 15% more than 10,000 eggs per gram. Six weeks after anthelmintic treatment, ova were not detectable in 44% of patients, there was at least a ten-fold fall in concentration in 7%, a five to nine-fold fall in 19%, a two to four-fold fall in 19% and no change in 11%. In the five patients receiving anthelmintic treatment weekly for 6 weeks, ova were not detectable from the 4th week onwards.

Serum IgE levels were measured before and 6 weeks after single dose anthelmintic treatment in forty-six sample pairs (Fig. 1). The geometric mean IgE level fell from 2310 to 1860 units per millilitre. This fall was highly significant ($P<0{\cdot}001$, paired t-test). Serum IgE

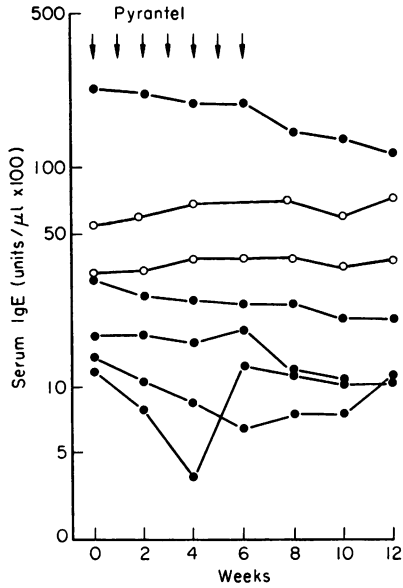

FIG. 2. Serum IgE levels measured fortnightly. (●) Subjects receiving anthelmintic treatment for 6 weeks. (○) Subjects not receiving specific therapy.

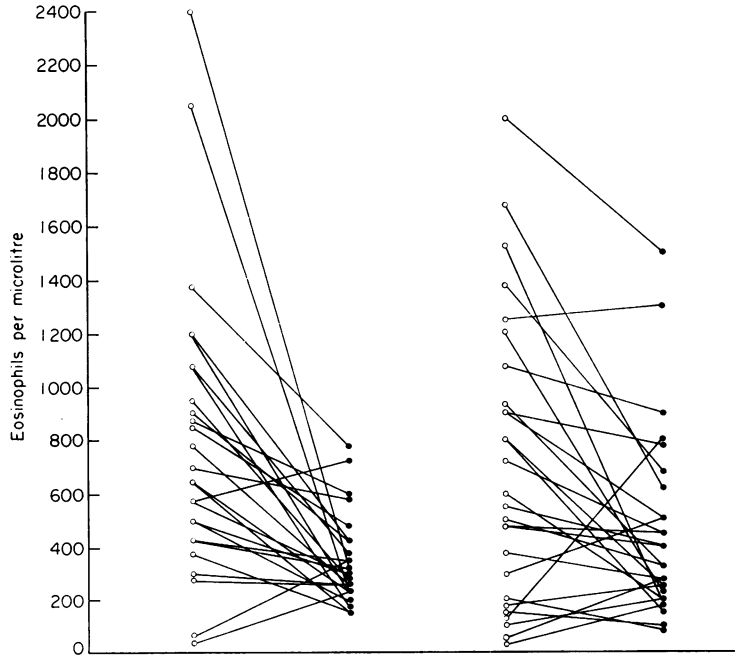

FIG. 3. Blood eosinophil levels before and 6 weeks after anthelmintic treatment in fifty-three subjects. (○) Pretreatment levels. (●) Post-treatment levels.

levels were measured before and after a 6-week interval in twenty subjects who did not receive anthelmintic therapy. No significant change occurred, the geometric mean IgE values being 2250 and 2420 units per millilitre respectively.

Serum was taken fortnightly from five subjects who received weekly anthelmintic treatment in the first 6 weeks, and from two control subjects (Fig. 2). A consistent decline in IgE levels over 3 months was seen in four subjects. The fifth showed an initial sharp fall, a rise at 6 weeks, then a slow decline. The two patients who did not receive specific therapy showed little change in IgE levels.

Blood eosinophil levels were measured in fifty-three sample pairs before, and 6 weeks after single dose anthelmintic treatment (Fig. 3). The mean level fell from 745 to 400 eosinophils per microlitre. This fall was highly significant ($P<0{\cdot}001$, paired t-test).

DISCUSSION

High serum IgE levels were found in Papua New Guinea Highlanders of the Goroka Subdistrict, an area of universal hookworm infestation. Anthelmintic treatment resulted in elimination of, or reduction in, worm burdens in most persons. This was associated with a fall in IgE levels 6 weeks after treatment.

Serial investigation over 3 months supports the view that IgE levels fall after anthelmintic therapy. The level fell steadily in four patients. The rise noted in one patient at 6 weeks, with no ova detectable in the stools, may reflect the larval stage of re-infection, as it has been shown that both the systemic larval phase and the gastro-intestinal adult phase stimulate IgE production in rats infected with *Nippostrongylus brasiliensis* (Jarrett & Stewart, 1973).

The fall in IgE levels parallels that seen after treatment of intestinal capillariasis, and after the acute phase of trichinosis. The fall may reflect a decrease in antigenic stimulus by elimination or reduction of the worm burden. It has been shown in animals that helminth infestation potentiates reaginic antibody responses to non-helminth antigens (Orr & Blair, 1969). Jarrett & Stewart (1973) have suggested that such responses may require maintenance by live worms over a period of time. The fall in IgE levels could also represent lower titres of non-helminth reaginic antibodies following removal of the worms.

Blood eosinophil levels also fell 6 weeks after anthelmintic treatment. This also is consistent with a lessened stimulus to eosinophilia after elimination or reduction of the worm burden.

These findings contrast with those reported by Biroum-Noerjasin (1973) in a Javanese population in which a rise in IgE levels after anthelmintic treatment was claimed. Several possibilities exist for this discrepancy. In that study, samples were taken before and 6 weeks after treatment, in only eight individuals, whereas we report forty-six subjects. Other results reported do not represent a comparison within the same group, but are from different individuals at varying periods after anthelmintic treatment. It is doubtful whether it is valid to compare such groups. Furthermore, ova were absent in only 17% of Javanese subjects compared with 44% of our subjects 6 weeks after treatment. It is possible that in the Javanese study either insufficient dose of anthelmintic was used or some patients did not take the drugs given.

The finding of a fall in IgE levels after anthelmintic treatment is satisfying in that it accords with the general observation in other immunoglobulin classes that antibody levels fall after removal of the antigenic stimulus.

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